We claim:

1. A method for identifying an agent which modulates the binding of a RGM to a Neogenin, the method comprising the steps of:

(a) forming a mixture comprising an isolated mammalian RGM and an isolated mammalian Neogenin;

(b) incubating said mixture in the presence of an agent; and

(c) detecting in the incubated mixture of step (b) the level of specific binding between said RGM and said Neogenin,

wherein a difference in the detected level of specific binding of said RGM to said Neogenin in the presence of said agent relative to the level of specific binding in the absence of said agent indicates that said agent modulates the binding of said RGM to said Neogenin.

2. A method for monitoring the interaction between an RGM and a Neogenin, the method comprising the steps of:

(a) contacting a first protein comprising the RGM with a second protein which comprises the Neogenin under conditions where a domain of the RGM binds to a domain of the Neogenin;

(b) determining the binding of the first protein to the second protein or second protein to the first protein.

3. A method for monitoring the interaction between a RGM and a Neogenin, the method comprising the steps of:

(a) contacting a fusion protein comprising an RGM domain with cells which express a Neogenin;

(b) detecting the fusion protein comprising the RGM domain which binds to the cells.

4. A method for monitoring the interaction between a RGM and a Neogenin, the method comprising the steps of:

(a) contacting a protein comprising a RGM domain with cells which express a polypeptide comprising the Neogenin;

(b) detecting the protein comprising the RGM domain which binds to the cells.

5. A method for monitoring the interaction between a RGM and a Neogenin, the method comprises the steps of:

(a) co-culturing in a matrix (a) embryonic nerve cells with (b) cells which have been transfected with an expression construct encoding the RGM and which express the Neogenin;

(b) adding to the cells an inhibitor of binding of the RGM and Neogenin;

(c) determining the axon outgrowth adjacent to the cells which express the RGM in the presence and absence of inhibitor.

6. A method for monitoring the interaction between a RGM and a Neogenin, the method comprising the steps of:

(a) culturing embryonic nerve cells under conditions in which they display growth cones;

(b) contacting the embryonic nerve cells with the RGM and an anti-Neogenin antibody;

(c) observing the effect of the antibody on the collapse of the growth cones.

7. A method according to any one of claims 1-6, wherein said RGM is a human RGM.

8. A method according to any one of claims 1-6, wherein said Neogenin is a human Neogenin.

9. A mixture comprising an isolated mammalian RGM and an isolated mammalian Neogenin.

10. A mixture according to claim 9, wherein said RGM is a human RGM or said Neogenin is human Neogenin.

11. A mixture according to claim 9, wherein said RGM is a human RGM and said Neogenin is human Neogenin.

12. A method of enhancing axon outgrowth comprising inhibiting the interaction between RGM and Neogenin.

13. A polypeptide portion of Neogenin useful for antagonizing the interaction between RGM and Neogenin.

14. An antibody preparation which specifically inhibits the interaction of a Neogenin protein and an RGM protein.

15. A use of an inhibitor capable of modulating the interaction between RGM and Neogenin in the prevention or treatment of a disease or condition associated with the degeneration or injury of vertebrate nervous tissue.

16. The use of claim 15 wherein said diseases or conditions associated with the degeneration or injury of vertebrate nervous tissue are selected from the group consisting of neurodegenerative diseases, nerve fiber injuries and disorders related to nerve fiber losses.

17. The use of claim 16, wherein said neurodegenerative disease is selected from the group consisting of motorneuronal diseases (MND), ALS, Alzheimer disease, Parkinsons disease, progressive bulbar palsy, progressive muscular atrophy, HIV-related dementia and spinal muscular atrophy(ies), Down's Syndrome, Huntington's Disease, Creutzfeldt-Jacob Disease, Gerstmann-Straeussler Syndrome, kuru, Scrapie, transmissible mink encephalopathy, other unknown prion diseases, multiple system atrophy, Riley-Day familial dysautonomia wherein said nerve fiber injuries are selected from the group consisting of spinal cord injury(ies), brain injuries related to raised intracranial pressure, trauma, secondary damage due to increased intracranial pressure, infection, infarction, exposure to toxic agents, malignancy and paraneoplastic syndromes and wherein said disorders related to nerve fiber losses are selected from the group consisting of paresis of nervus facials, nervus medianus, nervus ulnaris, nervus axillaris, nervus thoracicus longus, nervus radialis and for of other peripheral nerves.